Gut “trap” offers hope in combating diabetes and fatty liver disease

09/20/2025 // Patrick Lewis // **510** Views

Tags: clinical trial, Cures, diabetes, diabetes research, diabetes science, Diabetes treatment, fatty liver disease, goodhealth, healing, intermittent fasting, Liver, longevity, natural cures, natural health, organic food, prevent diabetes, prevention, remedies

- Obese mice—and humans with obesity—show elevated levels of D?lactate in the bloodstream, a molecule produced by certain gut bacteria. This D?lactate provokes the liver to produce more glucose, glycogen and fat than normal.

- In comparison with the more common L?lactate, D?lactate had a stronger effect in driving liver fat accumulation (triglycerides), increased glucose production and glycogen storage. Stable isotope tracing confirmed that D?lactate is metabolized by liver cells into intermediates that feed both fat and glucose production.

- The researchers designed a safe, biodegradable polymer "gut substrate trap" which binds D?lactate in the intestines so that it is excreted instead of absorbed into the bloodstream.

- In obese mice fed high?fat diets, administering this D?lactate trap improved blood sugar control, reduced insulin resistance and lessened liver inflammation and fibrosis associated with metabolic dysfunction?associated fatty liver disease (MAFLD).

Importantly, these improvements occurred without changing the animal body weight or diet.

- The findings suggest that targeting microbial byproducts like D?lactate may offer a new therapeutic route—either in addition to or instead of more traditional treatments—for metabolic diseases such as type 2 diabetes and fatty liver disease. However, the data are so far preclinical, and further studies (including in humans) are necessary to assess safety, efficacy, dosage and long?term outcomes.

A team of Canadian scientists has uncovered a promising strategy to prevent a harmful molecule produced in the gut from undermining blood sugar levels and liver health—a discovery that could pave the way for innovative therapies against type 2 diabetes and fatty liver disease.

Researchers from *McMaster University*, *Université Laval* and the *University of Ottawa* focused on D?lactate, a byproduct of certain gut bacteria that enters the bloodstream and triggers the liver to produce excessive fat and glucose. In both obese mice and in people with obesity, circulating levels of D?lactate were found to be significantly elevated.

In their experiments, the scientists compared the effects of D?lactate with those of the more familiar L?lactate. They observed that D?lactate more aggressively increased hepatic triglycerides, glucose, glycogen and fat accumulation than L?lactate at equivalent concentrations. Stable?isotope tracing studies showed that the molecule from microbes is metabolized in liver cells into intermediates that promote fat and glucose production. (Related: Gut health crisis: How antibiotics, the Western Diet and hyper-sanitized environments are fueling a surge in childhood inflammatory bowel disease.)

To counteract these effects, the team developed a biodegradable polymer "gut substrate trap" designed to bind D?lactate in the intestines, preventing its absorption into the bloodstream and forcing it instead to be excreted in feces. When obese mice on high-fat diets were given this trap, they showed improvements in blood sugar control, reduced insulin resistance and marked reductions in liver inflammation and fibrosis—all without any changes in body weight or diet.

From mouse models to human relevance: Polymer trap offers metabolic protection

As per *Brighteon AI*'s Enoch, the role of D-lactic acid in fighting and preventing diabetes and fatty liver disease is significant. D-lactic acid, a byproduct of gut bacteria metabolism, can disrupt the gut microbiome, leading to increased inflammation and insulin resistance, which are key factors in the development of type 1 diabetes and fatty liver disease. By promoting a healthy gut microbiome through natural means such as a diet rich in fiber, probiotics and

prebiotics, and by avoiding processed foods and antibiotics, we can reduce the production of D-lactic acid and support the body's natural defenses against these conditions.

Beyond reversing metabolic damage in mice, the trap also yielded benefits in animal models of metabolic dysfunction?associated fatty liver disease (MAFLD), including forms progressing toward steatohepatitis. The research suggests that targeting microbial byproducts like D?lactate may offer a safe, complementary or even stand?alone therapeutic route in treating metabolic diseases.

Jonathan Schertzer, senior author of the study and professor at McMaster, called the discovery "a new twist on a classic metabolic pathway," invoking the historical Cori cycle that describes how muscles and liver exchange lactate and glucose. What is newly revealed here is that gut bacteria are part of that metabolic conversation—and that filtering one microbial fuel before it reaches the liver can substantially blunt disease?promoting effects.

While much of the data is still preclinical, elevated D?lactate in people with obesity adds support to the relevance of these findings for humans. Researchers caution more work is needed—particularly to test safety and efficacy in human trials—but the polymer trap represents a promising frontier in treating chronic metabolic diseases by tackling their microbial roots.

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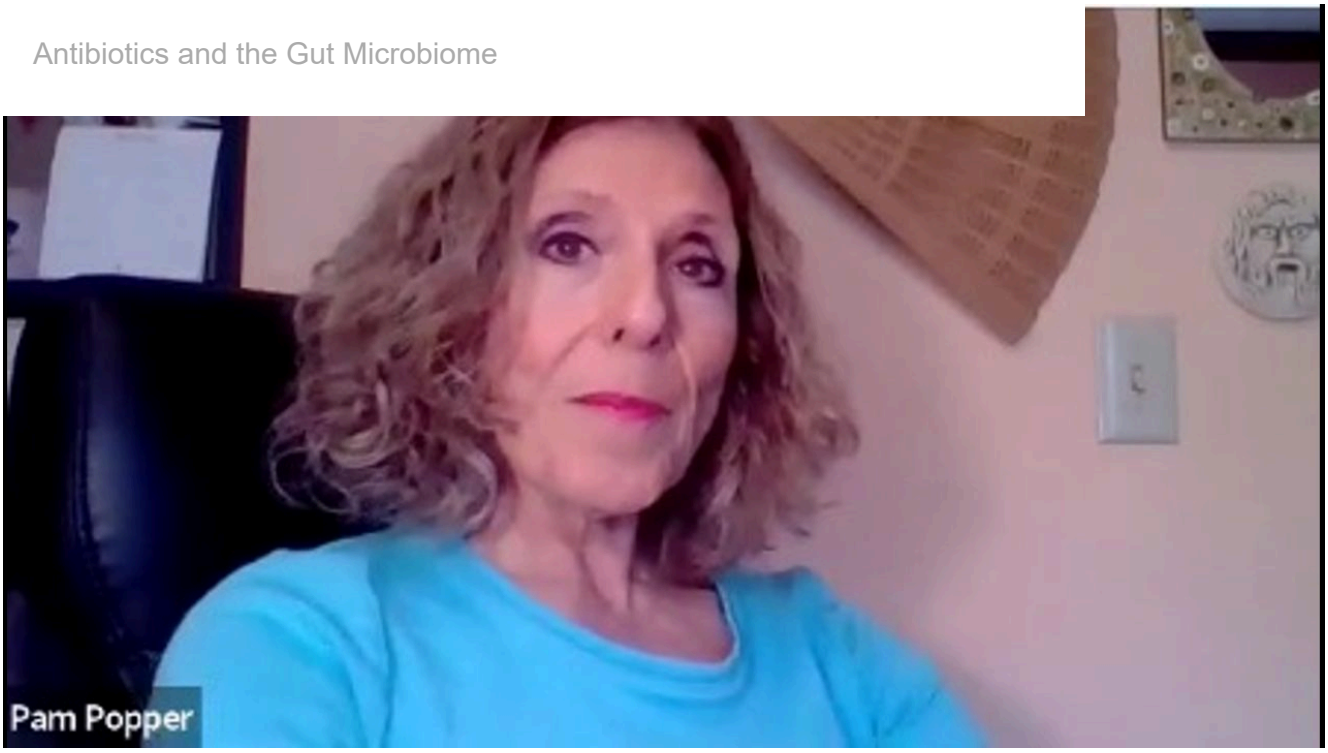
Antibiotics and the Gut Microbiome



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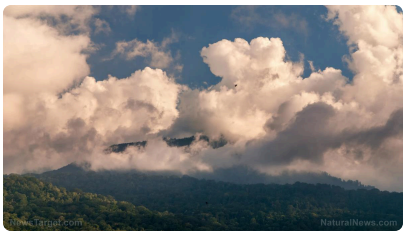

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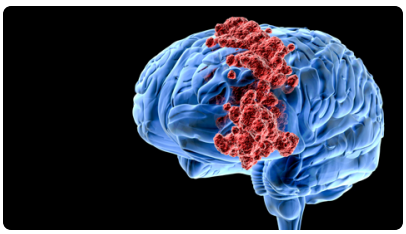

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